

Clinical Diagnosis and Prognosis of Ventricular Aneurysm

ATH. MOURDJINIS*, E. OLSEN, M. J. RAPHAEL, AND J. P. D. MOUNSEY

From the Departments of Medicine, Morbid Anatomy, and Radiology, and The Medical Research Council Cardiovascular Unit, Royal Postgraduate Medical School, London W.12

Where a full thickness infarct has occurred with replacement of myocardium by fibrous tissue, this inert portion of the ventricular wall cannot take part in concentric contraction and herniates outwards during ventricular systole. This localized disturbance of ventricular contraction after acute myocardial infarction is seen not only in large ventricular aneurysms but also with smaller full thickness infarcts (minor aneurysms) that have not caused a definite protrusion of the external surface of the heart. In this paper the range of localized disorders of ventricular contraction has been studied clinically, radiologically, at operation, and at necropsy. In diagnosis, special emphasis has been placed on abnormalities of the cardiac impulse, readily appreciated at the bedside, and on the information obtained from detailed fluoroscopy.

The prognosis after acute myocardial infarction depends not only on the possibility of a further infarct occurring, but also on the degree of damage that the infarct has inflicted on the ventricles as an efficient pump. The influence of major and minor aneurysms on prognosis following myocardial infarction has been examined.

SUBJECTS AND METHODS

The 39 patients studied fell into 2 main groups. There were 13 patients in whom the diagnosis of ventricular aneurysm was confirmed either at operation or at necropsy. The second group consisted of 26 patients in whom the diagnosis of ventricular aneurysm was made on clinical and radiological grounds alone. Of these 39 patients, 18 were diagnosed as having a ventricular aneurysm during admission to Hammersmith Hospital in a series of 112 consecutive patients treated in the Acute Coronary Care Unit (Shillingford and

Thomas, 1964). All 39 patients were examined clinically by one of us (A.M.) and the majority were seen in joint consultation. Of the 39 patients, 12 were hypertensive (blood pressure exceeding 140/90 mm.Hg on casual readings), one was diabetic, one was acromegalic, and one had multiple myelomatosis.

Definition of Ventricular Aneurysm. In this paper we have incorporated the concepts of Crawford (1943), Schlichter, Hellerstein and Katz (1954), and Luisada and Fleischner (1948) in our definition. Schlichter defined a cardiac aneurysm as a localized out-pouching of the cavity of a cardiac chamber, with or without outward bulging of the external surface. Luisada recognized anatomical and dynamic aneurysms, the latter showing paradoxical pulsation without any persisting bulge in the profile of the left ventricle. In both, a large transmural infarct gives rise to thinning of the left ventricular wall and consequent paradoxical pulsation. We have preferred to use the terms major and minor aneurysm. In a major aneurysm the localized out-pouching of the chamber cavity is accompanied by marked protrusion of the external surface, while in a minor aneurysm a suggestive bulge only may be seen.

Pathology. Post-mortem data were available in 7 patients; surgical specimens were obtained in 6 patients treated by ventriculoplasty for ventricular aneurysm. At necropsy the heart was first examined *in situ* to see whether any abnormalities in contour could be detected, after which it was removed in the routine manner. Water was then introduced into the left ventricle and gentle pressure applied to see whether outward bulging could be demonstrated. Post-mortem coronary arteriography was performed, the arteries having been injected with a mixture of Raybar cream (barium sulphate) and gelatine by the method of Harrison and Wood (1949). The heart was then dissected according to Lumb and Hardy (1964), and further radiographs taken. Next a vertical section was taken through the heart, the plane of section being chosen so that it passed through the summit of the aneurysm. This technique enabled

Received November 21, 1967.

* In receipt of a grant from the Greek State Scholarships Foundation, Athens, Greece.

a clear assessment to be made of the degree of myocardial thinning and its relation to the remaining healthy muscle and to the ventricular cavity. Representative blocks were subsequently selected and these were cut at 5μ thickness and stained with haematoxylin and eosin, Weigert's elastic van Gieson stain, and Martius Scarlet-Blue (Lendrum *et al.*, 1962). Blocks and sections from the surgical material were treated similarly. Dating of infarcts was based on criteria of Lodge-Patch (1951) and the age of appearances of thrombi was assessed by the criteria of Irniger (1962/3).

Cardiac Impulse. Detailed assessment was made of the nature of the cardiac impulse on clinical examination and in the impulse cardiogram. Since the major abnormality of the cardiac impulse was in ventricular systole, a faithful record of the palpable cardiac impulse could only be obtained in an *absolute* record of displacement, such as the impulse cardiogram which records total movement in relation to a fixed point in space (Beilin and Mounsey, 1962). Apex cardiography, which is a record of *relative* displacement of a point on the chest wall in relation to the immediately surrounding area, gives no constant pattern of the cardiac impulse in late systole. With this method the duration of the ventricular systolic impulse varies with the manual pressure with which the transducer is applied to the anterior chest wall and also with the total movement of the surrounding thoracic cage accompanying the heart beat (Mounsey, 1967). In recording absolute displacement with the impulse cardiograph a standard position was always adopted, with the patient reclining at 45° . Records taken in this position cannot be compared with those in the left lateral decubitus position, due to mediastinal shift and elevation of the left diaphragm, with consequent alteration of the heart's position in the thorax (Boicourt, Nagle, and Mounsey, 1965). Simultaneous phonocardiograms and electrocardiograms were recorded as reference tracings on a Cambridge multi-channel photographic recorder.

Electrocardiography. The electrocardiographic pattern in cardiac aneurysm comprises evidence of a transmural infarct with, in addition, persistent S-T segment deviation. Pathological Q waves in two or more adjacent chest leads, especially when monophasic, indicated the large size of an anterior or antero-lateral transmural infarct. Q waves in leads II, III, and aVF suggested a large transmural postero-inferior infarct. In anterior chest leads, persistent coved S-T segment elevation after healing of the infarct suggested an anterior or antero-lateral aneurysm. Persistent S-T segment depression in some precordial leads and S-T segment elevation in leads II, III, and aVF suggested a postero-inferior aneurysm. Although S-T segment elevation is suggestive of cardiac aneurysm, it is not diagnostic. Conversely, an aneurysm may be present in the absence of S-T segment deviation (Moyer and Hiller, 1951).

Radiology. In each patient plain chest films were obtained in postero-anterior and lateral projections.

Full distance 6 ft. films were taken except in 2 cases where only short distance portable films were feasible.

Fluoroscopy was carried out in all but 6 patients, using an image intensifier and television monitor system. Most patients were examined supine and in different phases of respiration. Meticulous fluoroscopy of the left ventricle in many projections is important, since abnormalities in contraction may only become obvious when they are shown in profile. The patients were examined straight and with varying degrees of obliquity, in both right and left anterior oblique views. In general, the postero-anterior projection demonstrates movements of the left cardiac border and apex, whereas in the right anterior oblique, movements of the anterior and inferior surface of the heart are better detected. In the left anterior oblique the postero-lateral contour is better visualized. To confirm our fluoroscopic findings, cineradiograms of the heart in appropriate projections were recorded in 25 of the 39 patients. These were taken with an image intensifier and a 35 mm. Ariflex camera at a speed of about 32 frames/sec.

Bi-plane roll-film angiocardiograms, with the left ventricle opacified by pulmonary artery injections of contrast medium, were performed in 5 patients, investigated prior to ventriculoplasty.

Clinical Course. Follow-up studies were made in every patient for between 1 and 13 years. Special note was taken of the nature of the cardiac impulse and of evidence of borderline left ventricular failure, troublesome angina, recurrent arrhythmias, and the occurrence of emboli. The clinical course was followed in this way of 33 patients medically treated. In a further 6 patients, under the care of Professor Goodwin, Dr. Celia Oakley, Mr. Cleland, and Professor Hugh Bentall, ventriculoplasty was carried out with excision of the aneurysmal wall, and their progress after operation was assessed.

RESULTS

(I) Patients with Major Ventricular Aneurysm

Ten patients had a large anterior ventricular aneurysm, of whom 6 were treated surgically by ventriculoplasty, while 4 eventually died and came to necropsy. The diagnosis of aneurysm was suggested clinically by the presence of an abnormal cardiac impulse, by localized protrusion or generalized enlargement of the left cardiac border in the chest x-ray film, with paradoxical pulsation on screening, and in 9 out of 10 patients by typical changes in the *electrocardiogram* (Table I, Fig. 1). Angiocardiography was carried out in 5 of the patients as a pre-operative investigation, and showed the exact site, shape, and abnormal contractions of the aneurysmal sac which was later removed during ventriculoplasty.

Clinical Course. The ages of the patients in this group varied between 38 and 72 years with an

TABLE I
ELECTROCARDIOGRAPHIC PATTERN IN PATIENTS WITH MAJOR VENTRICULAR ANEURYSM

Patient	Site of aneurysm	Associated infarcts	Monophasic Q waves	Other significant QRS changes	Coved S-T elevation	QRS width (sec.)
R.P.	* Anterior	—	V2-V6	—	V2-V7	0.12
J.P.	* Anterior	—	V1-V4	—	V2-V3	0.10
H.P.	* Antero-lateral	—	V2-V4	qR V5-V6	V2-V5	0.13
S.R.	* Anterior	—	V4R-V5	—	V2-V5	0.08
J.J.	* Antero-lateral	—	V4-V7	—	V4-V5	0.13
E.D.	* Anterior	—	V1-V6	—	V2-V6	0.09
M.N.	† Antero-lateral (calcified)	Anterior (subsequent)	V5-V6, I, VL	rS V7	—	0.15
T.S.	† Antero-septal	Inferior (previous)	V1-V5, II, VF, III	—	V2-V6	0.08
A.A.	† Anterior	Antero-septal (subsequent)	VL, V4	—	V4R-V4	0.14
J.G.	† Anterior	Posterior (previous)	V1-V2	rS V3-V6	V2-V7	0.12

* Confirmed at operation

† Confirmed at necropsy.

average of 54 years. One of the 10 patients was a woman. In the 6 patients treated by ventriculoplasty, the interval between the acute infarct giving rise to the aneurysm, and the operation, varied between 3 and 18 months. In all patients this was their first cardiac infarct. Four had continued ambulant for several weeks after the infarct. All 6 patients had been in borderline left ventricular failure with frequent angina of effort following the infarct. All 6 patients were in sinus rhythm, but paroxysmal arrhythmias were frequent, extra-

systoles, both ventricular and atrial, occurring in all. In addition, paroxysmal supraventricular tachycardia with 3 episodes of cardiac arrest occurred in 1 patient who had developed a large anterior aneurysm 3 weeks after his acute infarct and who was successfully treated by ventriculoplasty 11 weeks later. In spite of anticoagulant therapy, 1 patient had a systemic embolus originating from intracardiac thrombus, a finding noted at operation in 5 of the 6 patients. In another 3, the increasing size of the aneurysm suggested that there was danger

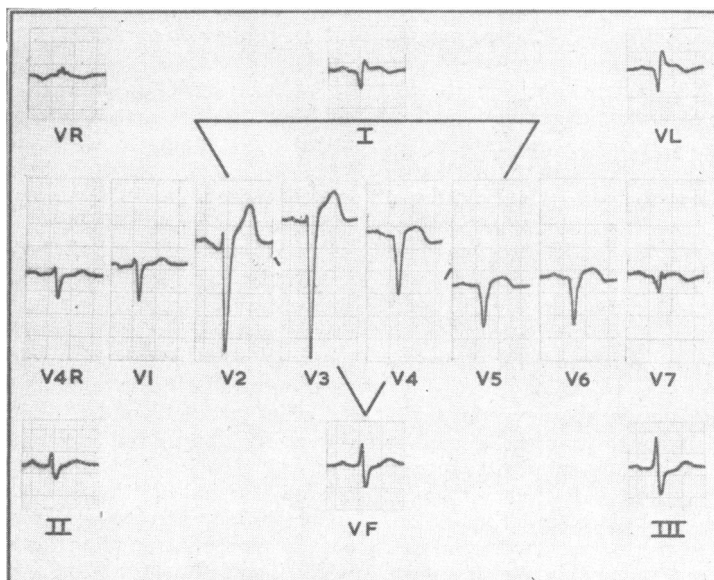


FIG. 1.—Electrocardiogram of major ventricular aneurysm 20 months after acute infarction. Widespread monophasic Q waves, persistent coved S-T elevation in V4-V7 and I and aVL. The aneurysm measured 15 × 10 cm. at operation and was antero-lateral in position.

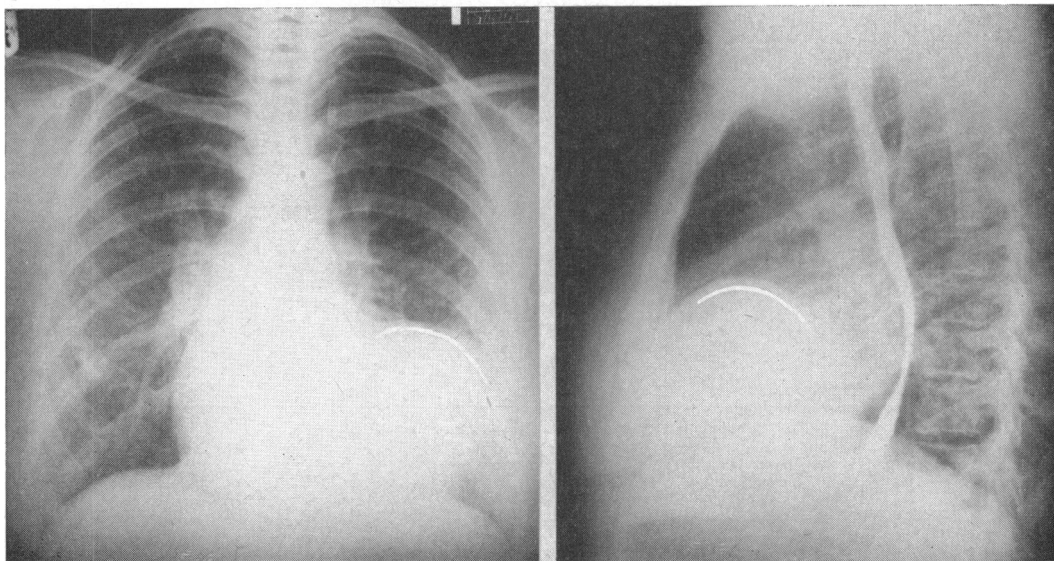


FIG. 2.—Postero-anterior and left lateral chest x-ray films of a patient with major ventricular aneurysm, showing curvilinear calcification (inked in) in wall of aneurysm. At necropsy, a saccular aneurysm was found to occupy most of the anterior wall of the left ventricle toward the apex.

of its rupturing. Of the 6 patients treated by operation, 5 have shown clinical improvement, with relief of troublesome angina of effort and disappearance of left ventricular failure. The sixth died of a further infarct that occurred a year after his operation.

Four patients were not treated surgically and eventually came to necropsy. All of these remained in sinus rhythm, apart from one who developed ventricular tachycardia terminally (*see below*). One patient had had a chronic aneurysm for about 10 years, while the others died within 1 to 20 months of developing the aneurysm. In the case of the chronic aneurysm, the patient had continued at work throughout the time of her first cardiac infarct, when the aneurysm was thought to have developed. Calcification of the aneurysm was first recognized 6 years before death (Fig. 2). She died of a further infarct, having had attacks of angina and borderline cardiac failure for 9 months previously.

The other 3 patients developed aneurysms within a few days of admission to hospital for cardiac infarction. Early admission to hospital had not been achieved and they had continued physical exertion for up to 2 days after the initial symptoms of infarction. Two of them had had a previous cardiac infarct. A pericardial rub was noted within the first week of the illness. The development of the aneurysm was recognized on the sudden appearance of a sustained cardiac impulse with typical electrocardio-

graphic and radiological changes and subsequent post-mortem confirmation. Two died during the same admission in hypotensive congestive heart failure. The third, who had had recurrent episodes of failure, lived 20 months and died during a third hospital admission in an episode of ventricular tachycardia, no evidence of a further fresh infarct being seen at necropsy.

Palpation and Auscultation. In all 10 patients, an abnormal *cardiac impulse* was felt. A lifting impulse was palpable above and internal to the apex beat, which was sustained in nature (Beilin and Mounsey, 1962; Mounsey, 1965). The site of greatest excursion (Table II) usually corresponded well with the centre of the underlying anterior ventricular aneurysm, as verified at thoracotomy or at necropsy. It usually extended over a wide area of the praecordium. Although we noted the appearance of an additional cardiac impulse internal to the apex beat, we did not consider the term "double cardiac impulse" clinically accurate in our cases (Libman and Sacks, 1927; Gorlin, Klein, and Sullivan, 1967). In our experience, a single abnormal impulse, greatest over the centre of the aneurysm, appeared to be propagated radially outwards over the surrounding area of the praecordium, which included the site of the apex beat (Mourdjinis *et al.*, 1966).

With one exception, the form of the impulse always showed the same abnormalities. It was composed

TABLE II
SITE OF CARDIAC IMPULSE IN MAJOR ANEURYSMS

Patient	Site of aneurysm	Apex beat		Ectopic maximum impulse	
		Left intercostal space	cm. from mid-sternal line	Left intercostal space	cm. from mid-sternal line
R.P.	Anterior	5th	12	3rd	10
J.P.	Anterior	5th	12	4th	9
H.P.	Antero-lateral	6th	17	6th	14.5
S.R.	Anterior	5th	12.5	4th	9
J.J.	Antero-lateral	5th	18	5th	11.5
E.D.	Anterior	5th	16	5th	10
M.N.	Antero-lateral	5th	6.5	4th	5
T.S.	Antero-septal	5th	10	4th	7
A.A.	Anterior	5th	8.5	5th	6
J.G.	Anterior	5th	12	4th	9

of 2 successive outward movements, easily recognizable on simultaneous inspection and palpation, combined with auscultation (Mounsey, 1966, 1967). Watching the movements of the stethoscope bell, while one is listening to the heart sounds, is a useful technique for this purpose. The first of the two impulses, which coincided with an atrial sound and immediately preceded the first heart sound, was an augmented atrial beat accompanying atrial systole. The second impulse closely following the first heart sound was a sustained, forceful, lifting impulse accompanying ventricular systole.

The impulse cardiogram confirmed these physical signs (Fig. 3). The atrial beat was abnormal in that it was of unusually large amplitude, since in health only a small outward movement accompanies atrial systole (Beilin and Mounsey, 1962). The ventricular ejection beat showed two abnormalities. First, it was of abnormally large amplitude and secondly it was sustained up to the time of the second heart sound. A sustained impulse of this type is never met in health where the impulse always returns to the baseline by the time of the second heart sound. When present it indicates

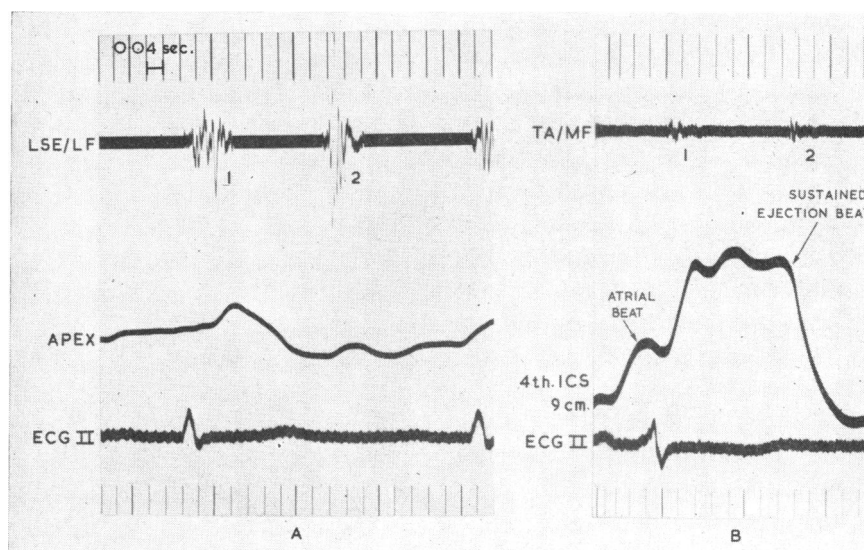


FIG. 3.—The impulse cardiogram in health and in major ventricular aneurysm. In health (A) the cardiac impulse (apex) moves outward in early systole but returns to baseline by the time of the second heart sound (2), with slight late systolic retraction. In cardiac aneurysm (B), the ectopic ventricular impulse (4th ICS, 9 cm. to left of mid-line) is of large amplitude and sustained throughout systole up to the time of the second heart sound (2). Preceding it is a large palpable atrial beat that accompanies atrial systole. Phonocardiogram: LSE/LF = left sternal edge, low frequency; TA/MF = tricuspid area, medium frequency. Electrocardiogram: lead II.

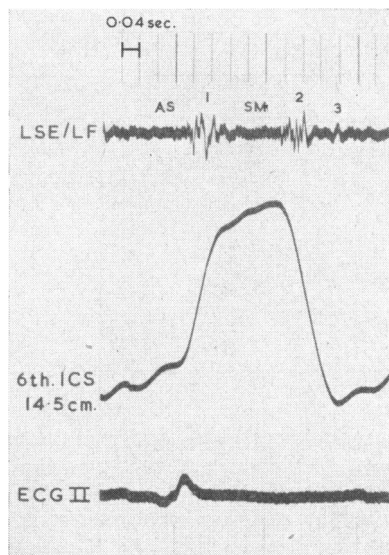


FIG. 4.—Sustained ventricular impulse with preceding atrial beat (6th ICS, 14.5 cm. to left of mid-line). Persistent third sound (3), atrial sound (AS), and apical systolic murmur (SM), in a patient with major ventricular aneurysm. The aneurysm was anterior measuring 5 × 7 cm. at operation and involved the anterior papillary muscle. Phonocardiogram: LSE/LF = left sternal edge, low frequency. Electrocardiogram, lead II.

either a ventricular aneurysm or more commonly ventricular hypertrophy (Beilin and Mounsey, 1962; Mourdjinis *et al.*, 1966).

In 1 patient a sustained cardiac impulse was not felt or recorded. This patient had heavy calcification of the wall of the aneurysm, and it is possible that this rendered the aneurysmal sac so rigid that paradoxical bulging of its walls during ventricular systole was prevented.

On auscultation all 10 patients had a third heart sound after the development of the aneurysm (Fig. 4). The third sound persisted throughout the period of follow-up, when patients were not in left ventricular failure. Thus, all 10 patients had both an atrial sound and a third heart sound, giving rise to a persistent quadruple rhythm. In the impulse cardiogram the third heart sound was accompanied by an augmented diastolic rapid inflow beat (Mounsey, 1966). An apical systolic murmur was heard in 4 patients, being loud in 1 and accompanied by a thrill at the apex, and relatively soft and blowing in the other 3 (Fig. 4). This sign suggested the presence of mitral incompetence and at operation all 4 patients were found to have papillary muscle involvement in the aneurysm (*see below*).

Radiology. In 8 of the 10 patients there was a clearly defined large local protrusion on the left

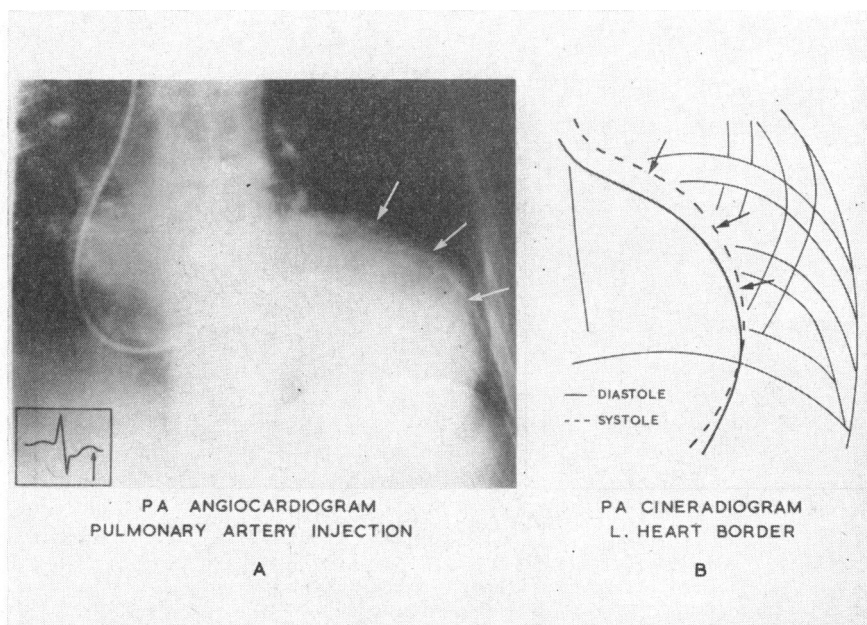


FIG. 5.—(A) Angiocardiogram of major ventricular aneurysm, antero-lateral in position (arrows) measuring 10 × 15 cm. at operation. (B) Tracings of pre-operative cineradiograms of the left cardiac border in systole (-----) and diastole (——), showing paradoxical pulsation (arrows) in region of aneurysm.

cardiac border (Fig. 2) which was calcified in 1 patient. In the other 2 patients whose anterior aneurysms were confirmed at necropsy, serial chest x-ray films showed a gradual increase in size of the left ventricle, but in neither was any localized bulge perceptible.

Fluoroscopy was carried out in all ambulant patients fit for this procedure, but could not be done in 2 patients who died during the episode of acute infarction giving rise to the aneurysm. Cine-radiography offered a useful permanent record of the pulsations. Fluoroscopy confirmed paradoxical pulsation in all 8 patients and was especially informative in 1 of the 2 patients with generalized left ventricular enlargement and no localized bulge. Here, gross paradoxical pulsation was seen over most of the left cardiac border in the postero-anterior view, thus confirming the diagnosis. Angiocardiograms in 5 of the 6 patients treated surgically confirmed the localized out-pouching of the left ventricular cavity and demonstrated marked thinning of the ventricular wall in the region of the aneurysm (Fig. 5).

Pathology. The aneurysmal wall was examined at necropsy in 4 patients and in surgical specimens in 6. Its thickness was shown to vary between 0.3 and 5.0 mm. The wall was seen to be composed almost entirely of fibrous tissue, with a few strands only of elastic tissue and practically no surviving cardiac muscle cells (Fig. 6). In addition, extensive calcification in the aneurysmal wall was seen in 1 patient. The site and extent of the aneurysm were examined at operation (Fig. 7) or at necropsy in every patient. The size of the aneurysm varied between 5 × 7 cm. and 10 × 15 cm., in diameter. The site of the aneurysm in all 10 patients was anterior, with extension laterally towards the septum

in 2 and towards the apex in 5. An area of pericarditis was noted over the aneurysm in all patients. In addition, mural thrombi were adherent to the wall of the aneurysm in 8 patients, and these were often of large size, measuring as much as 6 × 11 × 5.5 cm. in 1 patient. In 3 of the 6 patients treated by operation portions of the thrombi were noted to be friable and could be easily detached. Histology showed the thrombi to be laminated and to contain old and recent thrombotic elements. The anterior papillary muscle was involved in the aneurysm in 5 of the 10 patients, and, in addition, in 2 of these 5 patients the posterior papillary muscle was also implicated.

In 3 of the 4 patients who came to necropsy coronary arteriography was performed (Table III). This showed a large avascular area in the region of the aneurysm in the anterior wall of the left ventricle (Fig. 8). Extensive narrowing or occlusion of both right and left main coronary arteries was seen in each patient, with, in addition, recent occlusion of the left anterior descending artery in 2 of the patients.

Cardiac Impulse following Ventriculoplasty. The form of the cardiac impulse changed in all patients after operation. In 2 it reverted to a more normal pattern (Fig. 9), but in the remaining 4 some abnormalities remained, there being still an augmented atrial beat and a sustained ventricular ejection beat though of smaller amplitude. The explanation of these persisting abnormalities lay in the fact that with the larger ventricular aneurysms it was impossible to excise the whole aneurysm at the time of the ventriculoplasty, a rim of 1 cm. to 2 cm. being left. Parallel improvement in cardiac movement toward a more normal pattern was noted on fluoroscopy after operation.

TABLE III
POST-MORTEM CORONARY ARTERIOGRAPHY FINDINGS

Patient, age, sex	Heart weight (g.)	Aneurysm		Coronary arteries			
		Site and size	Type	Right	Left main	Left anterior descending	Circumflex
S.T. 66 M	570	Antero-septo-apical 8 × 6 cm.	Major	Old occlusion	Stenosed +++	Recent occlusion	Stenosed +++
A.A. 44 M	620	Anterior 8 × 10 cm.	Major	Old occlusion	Old occlusion	Small	Small
G.J. 72 M	470	Anterior 7 × 7 cm.	Major	Stenosed +++	Stenosed +	Stenosed +++ Recent occlusion	Stenosed +
W.J. 57 M	406	Posterior 7 × 6 cm.	Minor	Recent occlusion	Old occlusion	Old occlusion	Stenosed +
T.N. 64 M	510	Posterior (apex to mitral ring)	Minor	Stenosed +++	Stenosed +	Stenosed +++	Recent occlusion

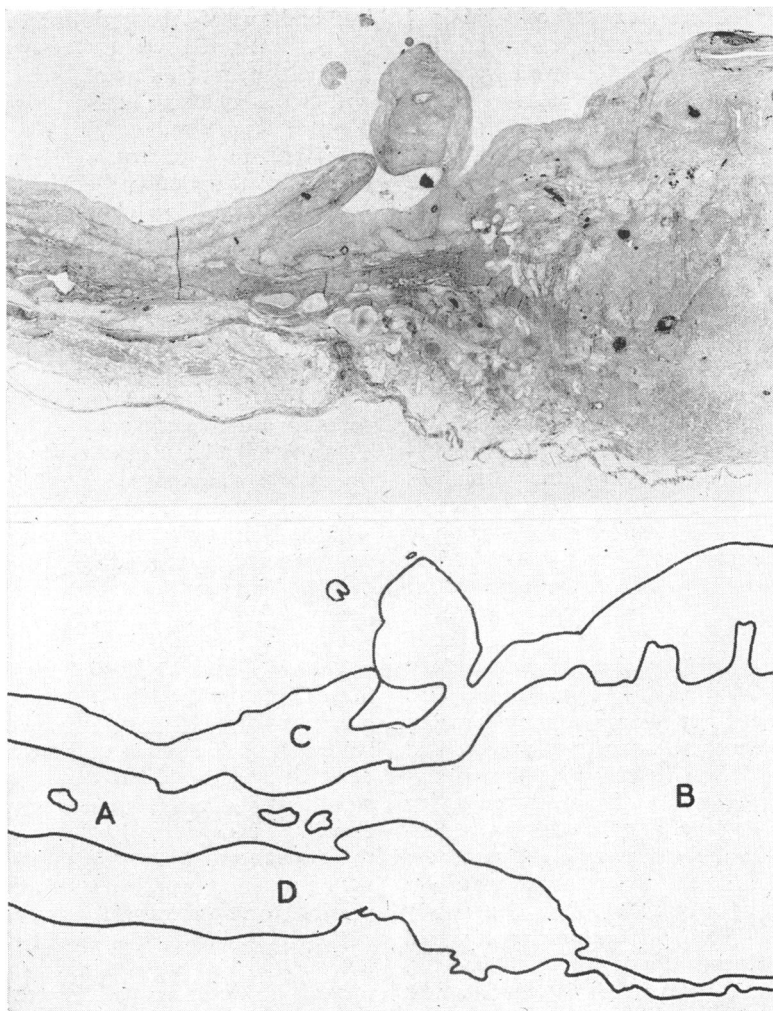


FIG. 6.—Microphotograph of section of major ventricular aneurysm showing junction of aneurysmal wall (A) and relatively normal cardiac muscle (B). Aneurysmal wall is thin, measuring 4 mm., and consists almost entirely of collagen tissue. The endocardial surface (C) is largely replaced by organized thrombus which projects into the lumen. Fatty deposits in epicardium (D). (Elastic van Gieson. $\times 6.4$.)

(2) Patients with Minor Ventricular Aneurysm

Twenty-nine patients who had sustained a transmural cardiac infarct showed localized paradoxical pulsation on fluoroscopy without marked protrusion of the external surface of the heart in the straight chest x-ray film. The site of the aneurysm was anterior in 22 patients and posterior in the remaining 7 patients. In the 3 patients who came to necropsy confirmation of the diagnosis of a minor ventricular aneurysm was obtained, while in the remaining 26 patients this diagnosis was made on clinical and radiological grounds.

Clinical Criteria for Diagnosis. Palpation and auscultation:

In every patient the cardiac impulse showed similar abnormalities to those already described in the presence of major ventricular aneurysm. An augmented atrial beat was followed by a sustained large ventricular ejection beat (Fig. 10). The site of this abnormal impulse varied with the site of the ventricular aneurysm. In 16 patients, in whom the aneurysm involved the anterior wall of the heart, the abnormal impulse was greater over an area of the praecordium internal to the apex beat. In another 5 patients, with anterior or antero-lateral involvement, the impulse was greatest at the



FIG. 7.—Operative exposure of an antero-lateral ventricular aneurysm (15 × 10 cm.).

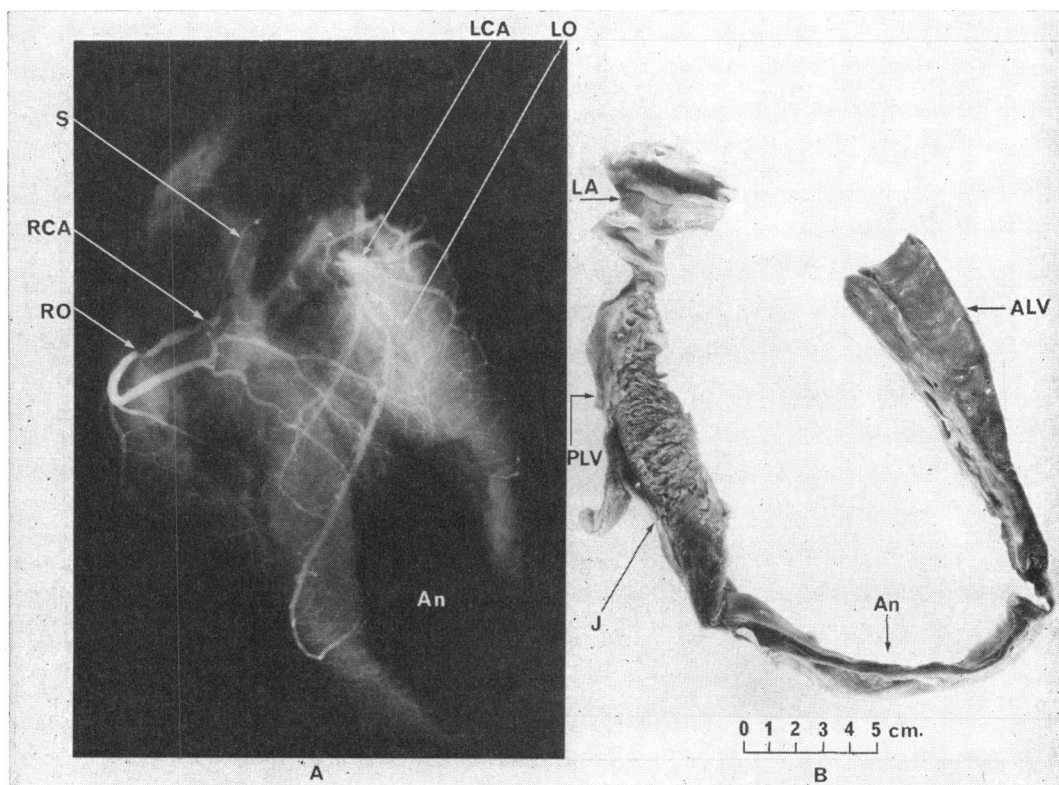


FIG. 8.—(A) Coronary arteriogram of intact heart. Right coronary artery (RCA) shows severe narrowing (RO) soon after beginning distal to branch to sino-aortic node (S). Left coronary artery (LCA) irregularly narrowed but medial branch shows total occlusion (LO). Dark area (An) shows extent of aneurysm. Extreme thinning of ventricular wall seen in tangential projection on left cardiac border, extending to apex. (B) Sagittal section through left ventricle and left atrium (LA) at thinnest portion of aneurysm (An). Junction of relatively normal posterior left ventricular wall (PLV) with aneurysm clearly shown (J). ALV = anterior left ventricular wall. Wall of aneurysm thinned to 1 mm. with superimposed lining thrombus.

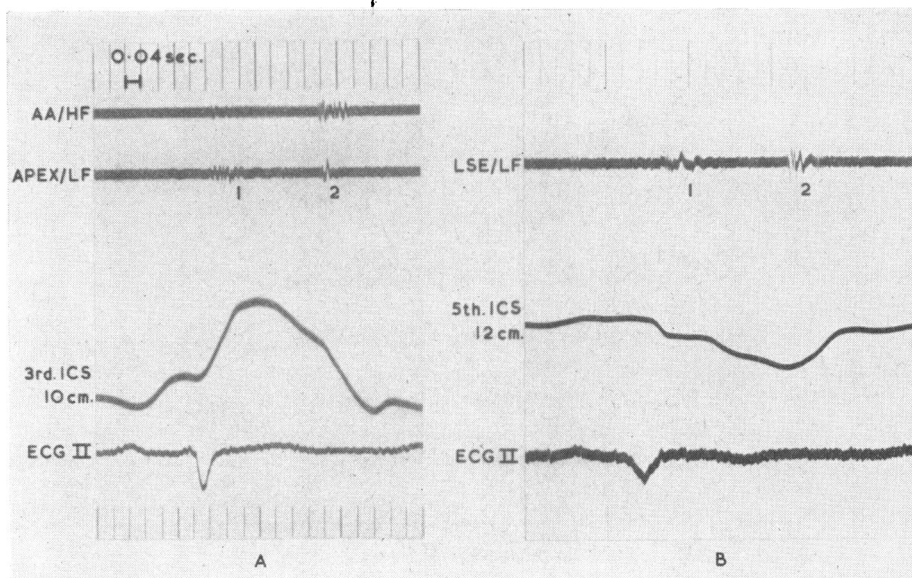


FIG. 9.—Reversion of sustained cardiac impulse to a more normal pattern after ventriculoplasty. (A) Pre-operative ectopic impulse (3rd ICS, 10 cm. to left of mid-line) sustained up to time of 2nd heart sound (2). (B) Post-operative apical impulse (5th ICS, 12 cm.) with late systolic retraction, from pleuro-pericardial adhesions. Phonocardiogram: AA/HF = aortic area, high frequency. APEX/LF = apex, low frequency. LSE/LF = left sternal edge, low frequency. Electrocardiogram: lead II.

apex. In the 7 patients with a postero-inferior aneurysm, however, an abnormal sustained cardiac impulse of smaller amplitude was most marked at the site of the apex beat and did not spread to involve the rest of the praecordium.

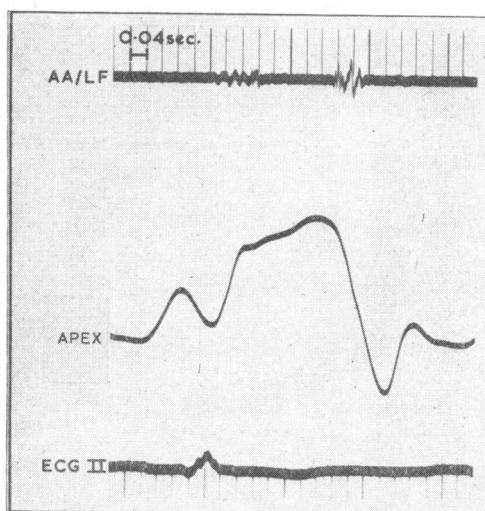


FIG. 10.—Impulse cardiogram in minor ventricular aneurysm. Large sustained apical cardiac impulse with preceding atrial beat; similar to that seen in major aneurysm. At necropsy, aneurysm was postero-lateral measuring 3 × 3 cm.

Twenty-four of these patients had a loud atrial sound and 18 had, in addition, a soft third heart sound constantly present over months or years. Five patients also had a blowing systolic murmur at the apex.

In 7 patients the development of the sustained cardiac impulse was observed during hospital admission for acute cardiac infarction: a normal cardiac impulse was noted on admission, but on the third or fourth day after the acute infarct, a sustained quality in the cardiac impulse was first observed. Although the amplitude of this impulse sometimes became less marked with the progression of convalescence, none the less its sustained nature remained and we have never seen the impulse revert to an entirely normal pattern.

Radiology. In contrast to the group of patients with major aneurysms, all of whom showed generalized cardiac enlargement, in only three-quarters of the patients with minor ventricular aneurysms was slight or moderate generalized enlargement seen. Thus, 15 of the 29 patients with minor aneurysms had cardiac enlargement, while a further 9 had slight enlargement only. In addition, in 19 patients bulging of the left upper border of the cardiac silhouette was seen in the postero-anterior chest x-ray film. Of these 19, a step-bulge was noted in 2, a rounded bulge in 11 (Fig. 11), and a suggestion of bulging in 6.

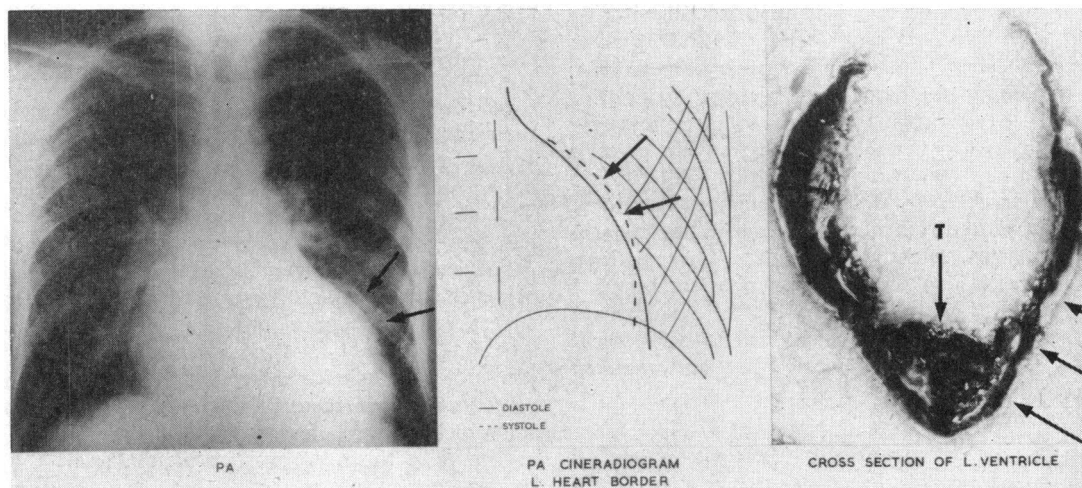


FIG. 11.—Minor postero-lateral ventricular aneurysm. Postero-anterior chest x-ray film (PA) showing rounded bulge (arrows). Cineradiogram tracing in systole (---) and diastole (—), demonstrating paradoxical pulsation (arrows). Cross-section of left ventricle through the plane of summit of aneurysm revealing extent (arrows) (5 × 3 cm.). Apex occupied by thrombus (T).

Fluoroscopy was the definitive radiological test for the presence of a minor cardiac aneurysm. This was carried out in 25 of the 29 patients, while in 2 of the remaining 4 patients necropsy proof of the aneurysm was eventually obtained. In every patient systolic expansile pulsation of a localized area of the heart border was seen. This pulsation was analysed by cineradiography in 18 patients. Systolic expansile pulsation was best seen in the right anterior oblique view in the 21 predominantly anterior aneurysms, while in the 4 postero-inferior aneurysms it was best seen in the left anterior oblique. In most patients in whom paradoxical pulsation was visible in an oblique view it was also visualized, though less clearly, in the postero-anterior view. In some patients paradoxical pulsation was visible in both oblique views as well as the postero-anterior view. In 2 patients with strictly anterior aneurysms, paradoxical pulsation was also seen anteriorly in the left lateral position.

Of special interest were 6 patients whose hearts were noted to enlarge while in hospital for acute cardiac infarction. In 2 of these 6, a suggestive localized fullness of the cardiac silhouette developed at the same time in the region of the aneurysm. Coincidental with these radiological changes was the development of an abnormal cardiac impulse, showing the sustained outward lift, of large amplitude, which we have already described as typical of cardiac aneurysm.

Electrocardiogram. This was compatible with the diagnosis of ventricular aneurysm in 27 of the

29 patients. In the case of the remaining 2 patients whose aneurysm was shown to be anterior on screening, one had right bundle-branch block and the other multiple previous infarcts. Twenty patients with an anterior aneurysm showed deep monophasic Q waves in at least 3 adjacent chest leads, while in the 7 postero-inferior aneurysms, large wide Q waves were seen in leads II, III, and aVF. Persistent S-T elevation was seen in anterior chest leads in the 20 patients with electrocardiographic evidence of an anterior aneurysm. With postero-inferior aneurysms persistent S-T depression was noted in lateral chest leads in 5 of the 7 patients; in addition, there was S-T elevation in leads III and aVF in 4. On these electrocardiographic criteria 10 of the aneurysms were anterior, 6 were antero-septal, 3 were antero-lateral, with spread posteriorly in a fourth. Two of the 7 postero-inferior aneurysms showed lateral involvement also, and in 2 the electrocardiogram was not diagnostic.

Clinical Course. There were 27 men and 2 women in this group of patients. Their ages varied between 38 and 79 at the time of the development of the aneurysm, with an average of 58 years. At least 6 of the patients were known to have continued active physical exertion for between 2 days and 2 weeks after the onset of the infarct giving rise to the aneurysm. Of the 29 patients, 25 are still alive: 4 died, 1 within the first year in congestive heart failure and 3 of further cardiac infarcts, 6, 8, and 13 years, respectively, after development of the

aneurysm. Recurrent left ventricular failure was a prominent symptom in 14 patients, and 9 of these required repeated hospital admissions for treatment; the remaining 15 patients have been free from heart failure. Severe angina of effort was a troublesome symptom in 6 patients, requiring frequent glyceryl trinitrate or long-acting vasodilators. All patients were in basic sinus rhythm, though extrasystoles, both atrial and ventricular, persisted for months after the acute infarct in 8 patients. Three patients had systemic emboli, 2 being peripheral and 1 cerebral, during the first month after development of the aneurysm. They happened to be 3 of the only 4 patients being treated by anticoagulant therapy in this group.

Pathology. Three of the 4 patients who died came to necropsy. In all, evidence of a minor ventricular aneurysm was seen, there being an obvious protrusion of the ventricular cavity at the site of the infarct within the thickness of the left ventricular wall and without localized external bulging. The whole heart section, embedded in paraffin and sliced through the area of the transmural infarct, enabled one to appreciate the disturbances of cardiac movement and contraction resulting from the minor ventricular aneurysm. In all 3 patients at least one-third of the muscular circumference of the left ventricle had been lost in this section and replaced by inert fibrous tissue (Fig. 11). Two of the aneurysms were posterior and of large size, one measuring 6×7 cm. The third was high lateral, extending posteriorly and of rather smaller size, measuring 3×3 cm. The thinnest portion of the aneurysmal wall varied between 3 and 5 mm. in these 3 patients. In only 1 of the 3 was intracardiac thrombus noted: histological examination of the aneurysmal wall showed it to be composed entirely of fibrous tissue with only a few strands of elastic tissue and practically no surviving myocardium.

Post-mortem coronary arteriography in 2 patients showed an avascular area in the posterior wall of the left ventricle at the site of the aneurysm (Table III); in 1 patient the anterior descending and right main coronary arteries were completely occluded, while in the other both the right and left main coronary arteries were stenosed with recent occlusion of the circumflex artery.

Infarction of the posterior papillary muscle was seen in all 3 patients. In addition, the anterior papillary muscle was infarcted in 1, where the aneurysm spread extensively onto the lateral wall of the heart. Clinical evidence of mitral incompetence with an apical systolic murmur was only found in 1 of these 3 patients.

(3) *Hypertension Complicating Ventricular Aneurysm*

Of the 39 patients with ventricular aneurysm, 12 were hypertensive, having blood pressures exceeding 140/90 mm.Hg at some time during their illness. In none did systemic hypertension appear to be the dominant disease clinically and only 5 patients needed mild hypotensive therapy. The electrocardiogram did not show evidence of left ventricular preponderance in any instance. All 12 patients, however, had electrocardiographic evidence of a large anterior transmural infarct with widespread, deep, monophasic Q waves, and, in addition, persistent S-T elevation supporting the diagnosis of ventricular aneurysm. Moderate generalized cardiac enlargement was present in 7 patients, slight or borderline enlargement in another 4, while in 1 patient the heart was normal in size. The configuration of the cardiac silhouette in the postero-anterior chest x-ray film showed the presence of an angulated bulge in 4, a rounded bulge in 5, and no clear bulge in 3. Fluoroscopy showed paradoxical pulsation in all 12 patients in a localized segment of the cardiac silhouette in the region of the aneurysm. Finally, all 12 patients had an abnormal cardiac impulse internal to the apex, of the typical sustained large amplitude seen in ventricular aneurysm. Although, therefore, some of the signs of ventricular aneurysm are difficult to differentiate from those of left ventricular hypertrophy from hypertension, careful attention to detail in these patients and to the general clinical picture established the diagnosis of ventricular aneurysm in addition to systemic hypertension.

(4) *Incidence and Prognosis of Ventricular Aneurysm following Myocardial Infarction*

Incidence. In order to assess the frequency of the development of a major or minor ventricular aneurysm after acute myocardial infarction, a study was made of 112 consecutive patients admitted to the Acute Coronary Care Unit at Hammersmith Hospital. The nature of their cardiac impulse was assessed and the electrocardiogram was scrutinized for evidence of ventricular aneurysm. Chest x-ray films were available in every patient and full fluoroscopic studies were carried out in varying oblique positions to detect paradoxical cardiac pulsation in 31 patients in whom cardiac aneurysm was suspected.

Eighteen of the 112 patients were found to have evidence of a ventricular aneurysm. In 3 a major aneurysm was present, while in the remaining 15 there was clinical, electrocardiographic, and radiological evidence of a minor aneurysm. In 2 of these 18 patients, however, the formation of the

aneurysm antedated the infarct for which they were admitted to hospital, having been present for between 5 and 7 years previously.

The incidence, therefore, of ventricular aneurysm in this series of 112 consecutive patients with acute cardiac infarction was 16 per cent. The incidence of the acute development of an aneurysm during this hospital admission was 14 per cent, the remaining 2 per cent of aneurysms being chronic and already present on admission.

Duration of Life after Onset of Ventricular Aneurysm. In all patients it was possible to make a fairly accurate estimation of the duration of the aneurysm following myocardial infarction (Table IV). Two patients died within the first month, and a third died during the remainder of the first year, giving a mortality rate of only 8 per cent in this year. Two further patients died during the second year, raising the mortality rate by the end of the second year to 13 per cent. Nine patients are known to have lived for longer than 5 years and 4 patients were known to have lived for longer than 9 years. The longest survival after aneurysm formation in this series was 13 years.

There were 4 late deaths in our series, occurring during the sixth, eighth, tenth, and thirteenth years, respectively. In each case the patient died of a further episode of cardiac infarction.

Prognosis in Patients with Major and Minor Aneurysms. In a smaller group of 13 patients consisting of 6 patients who died within 6 years and

TABLE IV
PROGNOSIS OF 39 PATIENTS WITH
VENTRICULAR ANEURYSM

Period of follow-up	No. of patients followed	Deaths in corresponding period
Within first month	39	2
Remainder of first year	37	1
Second year	24	2
Third year	18	—
Fourth year	9	—
Fifth year	9	—
Sixth year	8	1
Seventh year	6	—
Eighth year	4	1
Ninth year	4	—
Tenth year	2	1
Eleventh year	2	—
Twelfth year	2	—
Thirteenth year	1	1

7 survivors who were followed for 6 years, the prognosis of major and minor aneurysms was compared. Thus, 5 of the 7 patients alive after 6 years had minor aneurysms and only 2 had major aneurysms. Among the 6 who died, however, 4 had major aneurysms and 2 had minor aneurysms. Major aneurysms, therefore, appeared to carry a graver prognosis, particularly during the first 2 years (Fig. 12). It is also perhaps of significance that 1 of the 2 patients with the major aneurysm who survived 6 years had been treated by ventriculoplasty.

DISCUSSION

We feel that the use of the term "aneurysm" to include both those cases where there is protrusion of the external surface of the heart with out-pouching of the ventricular cavity as well as those

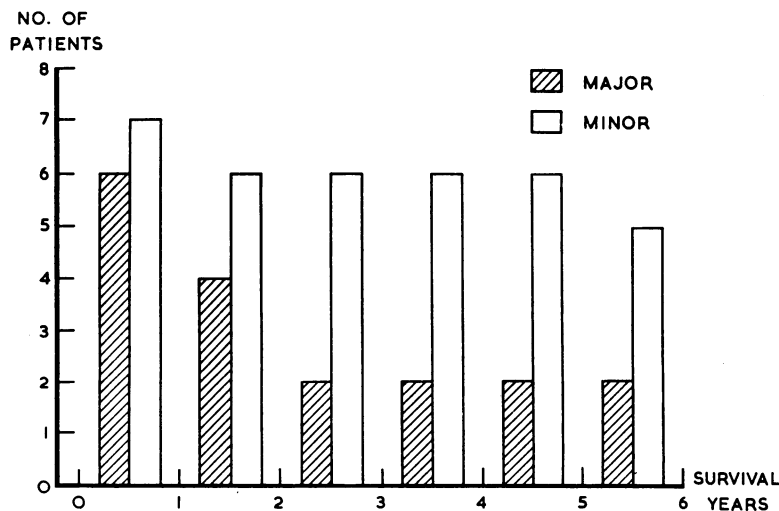


FIG. 12.—Prognosis in 13 patients with major and minor ventricular aneurysms.

where out-pouching of the cavity alone occurs is logical from the anatomical point of view (Schlichter *et al.*, 1954). Physiologically it is also acceptable since both these lesions result in localized abnormalities of ventricular contraction, with ballooning of the aneurysmal area during ventricular systole, giving rise to the sign of paradoxical pulsation on fluoroscopy (Braunbehrens, 1934; Luisada and Fleischner, 1948). In order to differentiate between lesions which do and do not produce localized external protrusion of the heart it seemed useful to employ the terms "major" and "minor" aneurysm, bearing in mind that even minor aneurysms are usually of considerable size with a diameter of 3-4 cm.

The recognition of the development of a ventricular aneurysm following acute myocardial infarction is usually easily made at the bedside. The appearance within the first few days after the infarct of a permanent sustained cardiac impulse is suggestive of a developing ventricular aneurysm. Screening confirms the presence of paradoxical pulsation in these patients. We noted in our patients that the excursion of the abnormal cardiac impulse often lessened with progression of convalescence, but its abnormally sustained nature always persisted as did paradoxical pulsation on screening. The evidence in our patients, therefore, pointed to the development of a ventricular aneurysm rather than to temporary acute myocardial ischaemia, with the transient and reversible abnormalities of ventricular contraction described by Tinsley Harrison (1959), in attacks of angina.

Persistence of a third heart sound in patients recovering from an acute cardiac infarct in the presence of other signs of left ventricular failure usually carries a poor prognosis: in ventricular aneurysm, however, a third heart sound may persist for many years after development of the aneurysm. It is possible, therefore, that the genesis of the third heart sound in ventricular aneurysm is in some way related to the aneurysm itself. The thin wall may act as a tambour, freely transmitting the intracardiac vibrations to the chest wall. All the patients were found to have an atrial sound in addition, the intensity of which may have been exaggerated by the same factors influencing the third heart sound. Another auscultatory physical sign met in one-quarter of the patients was an apical systolic murmur. On necropsy and operation evidence, we believe this usually reflects papillary muscle dysfunction, where these muscles originate from the aneurysmal wall.

Radiological recognition of major aneurysms presents no problem, since the aneurysm is immediately visible on the routine chest x-ray film. A

minor aneurysm, however, is likely to be missed, and fluoroscopy of the heart's movement to detect paradoxical pulsation is indispensable for diagnosis. Often the paradoxical pulsation can only be seen in one carefully selected projection after rotating the patient through various degrees of obliquity. In this way, posterior, inferior, or strictly anterior minor aneurysms may be visualized, in addition to those in the lateral region of the left ventricle which is more easily accessible radiologically. Only frank paradoxical pulsation was accepted as evidence of a minor aneurysm, though other abnormalities, such as localized diminution or absence of pulsation, were sometimes noted (Dack, 1955). Cineradiology provides a useful objective record of the paradoxical pulsation. Angiocardiography presents the most accurate method of diagnosis, affording detailed evidence of the site and size of the aneurysm, the thickness of the aneurysmal wall, and the form of its abnormal movement (Gorlin *et al.*, 1967). This investigation was performed in all but one of our patients treated by ventriculoplasty.

Since the diagnosis of ventricular aneurysm in this series was based primarily on necropsy and operation findings or on clinical and radiological evidence of local paradoxical pulsation, an opportunity was presented for verifying the accuracy of electrocardiographic signs of ventricular aneurysm. In general, we found close accordance between our diagnosis of aneurysm on other grounds and the electrocardiographic picture. Thus, of the 10 major aneurysms, all confirmed at operation or necropsy, in only 2 was the electrocardiogram not diagnostic. In 1 patient, left bundle-branch block obscured the electrocardiographic picture, while in the other, whose aneurysm was heavily calcified, there was no elevation of the S-T segment. In the other 8 patients in this group, widespread monophasic Q waves in adjacent praecordial leads were accompanied by S-T deviation having persisted for months or years after the development of the aneurysm. With 2 exceptions, a similar electrocardiographic pattern was seen in the group of 29 patients with minor ventricular aneurysm. Of the exceptions, 1 had right bundle-branch block, while in the other multiple previous cardiac infarcts obscured the electrocardiographic picture. The similarity of the electrocardiographic picture in major and minor aneurysm was to be expected, since the electrocardiogram probably reflects the extensive nature of the transmural infarct disturbing the equilibrium of the electrical forces in the heart as a whole (Moyer and Hiller, 1951), and is unlikely to be in any way directly related to external bulging of the aneurysmal wall.

In all 7 patients coming to necropsy, the diagnosis of aneurysm had been correctly made on clinical grounds. All the aneurysms, whether of the major or minor variety, were of large size. Thus, the smallest minor ventricular aneurysm measured 3×3 cm. in diameter, while the largest occupied the whole of the posterior and lateral walls of the left ventricle, producing generalized ventricular enlargement without localized protrusion. The largest major ventricular aneurysm measured 15×10 cm. in diameter. The site of the aneurysm was most frequently anterior, extending down to the apex, as reported by Parkinson, Bedford, and Thomson (1938), Dubnow, Burchell, and Titus (1965), and Gorlin *et al.* (1967). In only 7 of our 39 patients was the aneurysm postero-inferior in position. A point of interest was the frequency with which the papillary muscles were involved in the aneurysm: in all but one of the 7 necropsies one or more papillary muscles were shown to be infarcted, originating in the wall of the aneurysm. Intracardiac thrombi were a frequent finding, being present in all but 2 of the 13 patients coming to operation or to necropsy. The relatively high incidence of intracardiac thrombi lining the ventricular aneurysm was in accordance with the findings of others (Schlichter *et al.*, 1954).

Post-mortem arteriography showed a large avascular area in the region of the aneurysm which was anterior in 3 major aneurysms and posterior in 2 minor aneurysms. In all 5 patients severe arterial disease was present in both right and left coronary arteries with complete blockage of the main arterial supply to the area of the myocardium where the aneurysm had developed.

The incidence of ventricular aneurysm in ischaemic heart disease is naturally higher if both minor and major ventricular aneurysms are considered. In our 112 consecutive patients admitted to the Acute Coronary Care Unit, 18 were found to have ventricular aneurysms, of which 3 were major and 15 minor. The incidence of aneurysm was, therefore, 16 per cent and parallels fairly closely that recorded in 13 collected series quoted by Schlichter *et al.* (1954), in which the average incidence of ventricular aneurysm in instances of myocardial infarction found at necropsy was 15 per cent. Figures, however, differ widely in different series depending on the criteria accepted for the diagnosis of ventricular aneurysm. Thus, Dubnow *et al.* (1965) used the criteria of Edwards (1961), of a protrusion of a localized portion of the external aspect of the ventricle accompanied by a corresponding protrusion of the ventricular cavity: their reported incidence of ventricular aneurysm was only 3.5 per cent which, as might be expected, corresponded

fairly closely with the incidence of major ventricular aneurysm in our series (3%). Gorlin *et al.* (1967), on the other hand, using cineradiological criteria for diagnosis, found evidence of aneurysm in as many as 24 out of 100 cases of ischaemic heart disease. Their series, however, included cases where disturbances of contraction were seen at operation in a viable portion of cardiac muscle in which no morbid anatomical evidence of aneurysm would have been visible.

Douglas, Sferazza, and Marici (1962) gave an analysis of the survival after aneurysm formation in their 31 patients, where 11 deaths occurred in 5 months, giving a percentage mortality of 35, while 3 patients lived for 10 years or more, and the longest survival time was 15 years. In our series the early mortality rate was surprisingly low, being only 8 per cent in the first year. Our favourable prognosis in this early period is influenced by the fact that 4 patients with major aneurysm in intractable left heart failure before operation were successfully treated by ventriculoplasty within the first year, with resolution of symptoms, thus paralleling the good results reported by Bailey *et al.* (1958) and Chapman, Amad, and Cooley (1961). Another factor may have been the preponderance of minor aneurysms which in our series appeared to carry a better prognosis than major aneurysms, especially during the earlier years.

With one exception, where death occurred during a paroxysm of ventricular tachycardia, all the deaths after the end of the first year were due to fresh myocardial infarcts. Thus, the longest survival of 13 years was in a patient who sustained his first cardiac infarct at the age of 42, with radiological evidence at that time of aneurysm formation, and who lived for a further ten years before he had a second myocardial infarct: this was followed by 2 further cardiac infarcts, the last of which proved fatal. Penner and Peters (1946) emphasized that the prognosis of cardiac aneurysm may often be surprisingly good, with long survivals reported for as many as 17, 24, and even 40 years (White, 1933, 1937; Drake, 1940; Bland and White, 1941; Master *et al.*, 1954).

Recurrent episodes of left ventricular failure were a troublesome complication of aneurysm in our series. Of the 39 patients, 23 have had episodes of left ventricular failure after recovery from the acute infarct and have required digitalization and long-term diuretic treatment, with, in 10 instances, one or more subsequent hospital admissions. Of the 3 early deaths within the first year, all died in congestive heart failure, while a further 4 patients were treated by ventriculoplasty with permanent resolution of heart failure. Our ex-

perience of the frequency of heart failure in ventricular aneurysm is paralleled by the findings of others (Schlichter *et al.*, 1954; Chapman *et al.*, 1961).

Systemic emboli were a rare complication of aneurysm in our series, in spite of the demonstration of intracardiac thrombus lining the aneurysm in all but 2 of the patients in whom the heart was examined either at operation or at necropsy. Anticoagulant therapy was not given routinely in our series of patients, nor do our figures suggest that it was effective in preventing this complication. Thus, of the 10 patients on anticoagulant therapy, 3 developed systemic emboli, while of the 28 patients not on anticoagulant therapy only 1 developed an embolus.

Serious arrhythmias after resolution of the acute infarct were seen in only 2 patients in our group. One had episodes of supraventricular tachycardia shortly before successful ventriculoplasty, and the other developed paroxysmal ventricular tachycardia 2 years after the development of his aneurysm and died in an episode of this arrhythmia. Although rupture of the heart is a recognized complication of ventricular aneurysm (Abrams *et al.*, 1963), we did not meet this complication.

The results in our series suggest that the onset of intractable heart failure after the development of a cardiac aneurysm in ischaemic heart disease is a reliable indication for surgical treatment by ventriculoplasty, provided the remainder of the myocardium is relatively sound. The outlook for these patients without operation is poor, while with it the outcome has been highly satisfactory. If, however, cardiac function adjusts satisfactorily to the haemodynamic disabilities imposed by the aneurysm, the long-term prognosis is relatively good and appears to be governed primarily by the state of the remainder of the coronary arterial tree and the likelihood of further infarcts occurring. In these patients, therefore, in contradistinction to those in heart failure, there seems no clear indication for ventriculoplasty.

SUMMARY

Thirty-nine patients with ventricular aneurysm following myocardial infarction have been studied. The diagnosis was based on operation or necropsy findings in 13 of them; in the remainder the clinical diagnosis was made on the abnormal cardiac impulse recorded in the impulse cardiogram, on the electrocardiographic pattern, and on the radiological appearances of the heart studied with fluoroscopy and cineradiography.

Ventricular aneurysms were divided into major and minor types. In both a large transmural

myocardial infarct had occurred. While in major aneurysms external bulging of the cardiac silhouette was seen, in minor ventricular aneurysms, paradoxical pulsation of a localized segment of the cardiac border was present with little or no external bulging of the cardiac silhouette. The importance of palpation of the cardiac impulse and of detailed fluoroscopy in diagnosis is emphasized.

The incidence of major and minor aneurysms following cardiac infarction was relatively high: 16 per cent. Their natural history, prognosis, and treatment are discussed.

We would like to thank Professor H. H. Bentall, Professor C. C. Booth, Mr. W. P. Cleland, Dr. C. L. Cope, Dr. C. M. Fletcher, Professor Russell Fraser, Professor J. F. Goodwin, Dr. E. Montuschi, Dr. Celia Oakley, and Professor J. P. Shillingford for allowing us to include in our study patients under their care. We are also grateful to Mr. Peter Burgess, Miss Jean Powell, Mr. C. Brecknell, Mr. W. Brackenbury, and Mr. P. Stinton for technical assistance.

REFERENCES

- Abrams, D. L., Edelist, A., Luria, M. H., and Miller, A. J. (1963). Ventricular aneurysm. *Circulation*, **27**, 164.
- Bailey, C. P., Bolton, H. E., Nichols, H., and Gilman, R. A. (1958). Ventriculoplasty for cardiac aneurysm. *J. thorac. Surg.*, **35**, 37.
- Beilin, L., and Mounsey, J. P. D. (1962). The left ventricular impulse in hypertensive heart disease. *Brit. Heart J.*, **24**, 409.
- Bland, E. F., and White, P. D. (1941). Coronary thrombosis (with myocardial infarction); ten years later. *J. Amer. med. Ass.*, **117**, 1171.
- Boicourt, O. W., Nagle, R. E., and Mounsey, J. P. D. (1965). The clinical significance of systolic retraction of the apical impulse. *Brit. Heart J.*, **27**, 379.
- Braunbehrens, v. H. (1934). Die Herzmuskelschwiele und das Herzwandaneurysma. (Kongressheft.) *Fortschr. Röntgenstr.*, **50**, 15.
- Chapman, D. W., Amad, K., and Cooley, D. A. (1961). Ventricular aneurysm. *Amer. J. Cardiol.*, **8**, 633.
- Crawford, J. H. (1943). Aneurysm of the heart. *Arch. intern. Med.*, **71**, 502.
- Dack, S. (1955). The ventricular pulsations in myocardial infarction; a fluoroscopic and kymographic study. *Dis. Chest*, **27**, 282.
- Douglas, A. H., Sferazza, J., and Marici, F. (1962). Natural history of aneurysm of ventricle. *N.Y. St. J. Med.*, **62**, 209.
- Drake, E. H. (1940). Long survival following coronary thrombosis. *Amer. Heart J.*, **20**, 634.
- Dubnow, M. H., Burchell, H. B., and Titus, J. L. (1965). Postinfarction ventricular aneurysm. *Amer. Heart J.*, **70**, 753.
- Edwards, J. E. (1961). *An Atlas of Acquired Diseases of the Heart and Great Vessels*, Vol. II, p. 615. W. B. Saunders, Philadelphia.
- Gorlin, R., Klein, M. D., and Sullivan, J. M. (1967). Prospective correlative study of ventricular aneurysm. Mechanistic concept and clinical recognition. *Amer. J. Med.*, **42**, 512.

- Harrison, C. V., and Wood, P. (1949). Hypertensive and ischaemic heart disease: a comparative clinical and pathological study. *Brit. Heart J.*, 11, 205.
- Harrison, T. R. (1959). Some clinical and physiologic aspects of angina pectoris. *Bull. Johns Hopk. Hosp.*, 104, 275.
- Irniger, W. (1962/3). Histologische Alterbestimmung von Thrombosen und Embolien. *Virchows Arch. path. Anat.*, 336, 220.
- Lendrum, A. C., Fraser, D. S., Slidders, W., and Henderson, R. (1962). Studies on the character and staining of fibrin. *J. clin. Path.*, 15, 401.
- Libman, E., and Sacks, B. (1927). A case illustrating the leucocytosis of progressive myocardial necrosis following coronary artery thrombosis. *Amer. Heart J.*, 2, 321.
- Lodge-Patch, I. (1951). Ageing of cardiac infarcts and its influence on cardiac rupture. *Brit. Heart J.*, 13, 37.
- Luisada, A. A., and Fleischner, F. G. (1948). Studies of fluoro-cardiography: tracings of the left ventricle in myocardial infarction. *Acta cardiol. (Brux.)*, 3, 308.
- Lumb, G., and Hardy, L. B. (1964). Technique for dissection and perfusion of the heart. *Arch. Path.*, 77, 233.
- Master, A. M., Jaffe, H. L., Teich, E. M., and Brinberg, L. (1954). Survival and rehabilitation after coronary occlusion. *J. Amer. med. Ass.*, 156, 1552.
- Mounsey, J. P. D. (1965). The cardiac impulse during ventricular systole. *Amer. Heart J.*, 70, 279.
- (1966). The impulse cardiogram and the phonocardiogram. *Cardiologia (Basel)*, 48, 203.
- (1967). Inspection and palpation of the cardiac impulse. *Progr. cardiovasc. Dis.*, 10, 187.
- Mourdjinis, Ath., Olsen, E., Taubman, J., and Mounsey, J. P. D. (1966). Cardiac impulse in ventricular aneurysm. *Brit. Heart J.*, 28, 430.
- Moyer, J. B., and Hiller, G. I. (1951). Cardiac aneurysm: clinical and electrocardiographic analysis. *Amer. Heart J.*, 41, 340.
- Parkinson, J., Bedford, D. E., and Thomson, W. A. R. (1938). Cardiac aneurysm. *Quart. J. Med.*, 7, 455.
- Penner, S. L., and Peters, C. M. (1946). Longevity with ventricular aneurysm. *New Engl. J. Med.*, 234, 523.
- Schlichter, J., Hellerstein, H. K., and Katz, L. N. (1954). Aneurysm of the heart: A correlative study of one hundred and two proved cases. *Medicine (Baltimore)*, 33, 43.
- Shillingford, J. P., and Thomas, M. (1964). Organisation of unit for intensive care and investigation of patients with acute myocardial infarction. *Lancet*, 2, 1113.
- White, P. D. (1933). Longevity after coronary thrombosis. *J. Amer. med. Ass.*, 100, 233.
- (1937). A new record in longevity after coronary thrombosis. *J. Amer. med. Ass.*, 108, 1796.